# Asynchronism in Mitral Stenosis

By C. C. GIBBES, M.D., M.R.C.P. Lond.


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## ASYNCHRONISM IN MITRAL STENOSIS.

BY C. C. GIBBES, M.D., M.R.C.P. LOND.,
Physician and Pathologist to the Hospital for Diseases of the Heart.

Mitral stenosis has provoked more discussion than any other form of valvular heart disease, yet in spite of it all the mechanism of the disease is, to say the least, rather involved; each theory brought forward as to the cause of the presystolic murmur being founded on a totally different conception of the heart's action in this disease.

Some authorities still support the theory enunciated by Ormerod and Barclay, that the murmur characteristic of this disease is not presystolic but systolic in rhythm, and caused by the systole of the left ventricle. The majority, however, have accepted the "Gairdner" theory, viz., that the murmur is postdiastolic and produced by the flow of blood from the left auricle into the left ventricle. Dr. Brockbank has brought forward a modification of the former theory, but has adhered to the principle that the murmur is systolic. There have been therefore practically only two theories before the profession; these we style for short the "Barclay" and the "Gairdner."

The theory I have ventured to bring forward elsewhere agrees in the main with the Gairdner mechanism, my opinion being that the presystolic murmur is composed of three parts:

1. Audible right ventricular muscle vibrations. In consequence of the two ventricles not acting synchronously a portion of the right ventricular systole takes place, and its muscle vibrations are heard while the left ventricle is in diastole.

2. A murmur caused by the flow of blood from the left auricle into the left ventricle, and called by Professor Gairdner the auriculo-systolic murmur.

3. The slapping first sound. This forms part and parcel of the murmur and ought to be so considered.

The discussion that was carried on in *The Lancet* during the years 1872, 1887, and 1889 satisfied the majority of the profession that the Barclay theory was untenable; but while disproving that theory Professor Gairdner's supporters failed to answer several pertinent questions asked by their opponents, nor have they been satisfactorily answered by subsequent writers on this subject. The majority of the arguments brought forward in favor of the Gairdner theory have been founded on tracings of the cardiograph, although Roy and Adami have proved that such tracings cannot be relied on. To show the feebleness of these arguments it will be sufficient to

quote briefly from a recent article on mitral stenosis (Allbutt's System of Medicine, vol. v). Referring to cardiograms obtained by placing the tambour over the apex beat, the writer tells us on page 1015 that "the pen of the cardiograph is guided by the apex of the left ventricle; the record of the auricular systole is written by an impulse communicated from the auricle to the ventricle." He therefore appears to have overlooked the fact that the apex of the heart, in pure mitral stenosis, is formed by the right ventricle alone, and in that disease the left ventricle is not in contact with the chest wall at all. In fact, the evidence is manufactured regardless of morbid anatomy, no distinction being drawn between tracings taken in pure mitral stenosis and in double mitral disease.

The special utility in these tracings consists in their being able to prove anything you wish, the same ones having been used to prove and disprove both the Barclay and the Gairdner theories; and I might with equal justice use them in support of my own theory, to prove that the tambour placed over the apex beat receives vibrations from the right ventricle, and consequently the tracings show that the right ventricle contracts before the left. It is unnecessary to enlarge on this subject, as the above facts are, I think, sufficient to demonstrate the true value of cardiographic evidence.

The object of my present paper is to discuss briefly the mechanism of mitral stenosis and to show that the special characters of the presystolic murmur are due to reduplication of the first sound, in consequence of the two sides of the heart not acting synchronously.

We have in pure mitral stenosis a slowly progressing *constriction* of the mitral orifice, which renders the process of filling the left ventricle more and more difficult as the disease progresses. This slowness of onset enables the heart to gradually adapt itself to these altering conditions, and we find that the difficulties are met by the auriculo-systolic blood current becoming accelerated and the diastole of the left ventricle occupying a larger portion of the cardiac cycle than in the normal heart. In the early stages hypertrophy of the left auricle and an increase in the suction power of the left ventricle are able to effect this increased velocity, but as the disease advances they prove insufficient, and the right ventricle and pulmonary arteries become hypertrophied and gradually take on the work, the left auricle being often incapable of any contraction. The right ventricle thus aids or assumes the functions of the left auricle. Now to do this, and at the same time to have a purely ventricular rhythm, would be an impossibility; consequently, to meet the requirements of the circulation the systole of the right ventricle is prolonged, the

early portion of it being auricular in rhythm and occurring while the left ventricle is in diastole. In other words, the two sides of the heart do not act synchronously, and I contend that the circulation could not be carried on in this disease if they did. This is borne out as follows:

1. The conditions of interventricular tension on the right and left sides of the heart. We know that we have low tension in the left ventricle and high tension in the right. We know that while the right ventricle is engorged and has a difficulty in getting rid of its contents, the left suffers from a process of starvation, and nature's efforts are greatly taxed to supply it with sufficient blood to carry on the circulation. Consequently the left ventricle has a prolonged diastole in order that it may become sufficiently full to meet the requirements of the circulation, and being insufficiently filled the systole is short and sudden. On the other hand, the right ventricle has an entirely different condition of affairs to deal with; it is always too full, the diastole is curtailed, and the systole is prolonged, the latter commencing while the left ventricle is still in diastole. Thus the right ventricle assumes not only the functions but also the rhythm of the left auricle.

2. Reduplication of the second sound. This is almost invariably present as long as the heart is properly compensated, its presence depending on both the aortic and pulmonary second sounds being audible. According to some authorities the reduplication is frequently absent at the base and yet present at the apex; this, however, is not my experience. We cannot expect to hear the same interval between the reduplicated sounds when we listen over the site of the valves, where the sound waves originate, as we do at the apex, where the waves have had a greater amplitude; consequently, while at the apex we hear a distinct interval, at the base the aortic and pulmonary second sounds can be heard side by side without any interval; they, in fact, are often mistaken for one sound, but careful stethoscopy will reveal the want of perfect synchronism. Cases do occur, however, where the reduplication is *really* absent at the base and present at the apex, but I believe they are very rare. In propounding theories to account for this, it appears to have been forgotten that in health the second aortic sound is always heard louder to the left of the apex beat than over the site of the valves, and that in mitral stenosis, while both the base and the apex are pushed further back from the surface of the chest, by the engorged left auricle and right heart, the former causes the base to be displaced to a greater extent than the apex.

We have therefore two reasons why the second aortic sound should be lost at the site of the valves before it is at the apex.

3. Reduplication of the first sound.

The presystolic murmur may be said to be fairly characteristic of mitral stenosis, although it is by no means pathognomonic, but it must be remembered that it is not a mere murmur that we have to deal with, but a rhythm of which the murmur forms only a part.

In typical cases we hear on auscultating the apex:

1. A slapping first sound, and provided the aortic second sound is audible, a reduplicated second sound.

2. A shortened systole and a prolonged diastole.

3. A harsh postdiastolic murmur, crescendo in character, and ending abruptly with the first sound, of which it forms an integral part. The murmur varies in length and may extend backwards to the second sound; as the disease advances it frequently varies, but these variations follow certain well marked lines, and assist us in forming a prognosis and prescribing treatment. The *Lancet* discussions on the origin of the presystolic murmur originated in a case of mitral stenosis with bradycardia that came under Dr. Barclay's care, and concerning which he wrote as follows: "The contraction of the heart could be felt beween the ribs during the pause, prior to the first sound and while the presystolic murmur was audible to the ear" (*Lancet*, Feb. 3, 1872, p. 284). I have recently reported a similar case (*Clinical Journal*, Dec. 7, 1899), in which I found that by laying my finger along the fifth left intercostal space I was able to feel the ventricular systolic wave run along my finger and end with the apex beat.

The character of the movement has evidently been quite misunderstood; it is not a "lifting" movement as suggested by Dr. Dickinson (*Lancet*, Oct. 19, 1889, p. 782), and cannot be satisfactorily explained by the hypertrophied left auricle causing "the propulsion of the heart's apex against the wall of the chest" (Sansom, Allbutt's System of Medicine, vol. v, p. 1015). Moreover, it did not in the least resemble the undulations of a dilated heart or the diffuse impulse of dilatation with hypertrophy. On combining auscultation with palpation, I found that it occupied more than half the entire diastolic period. I have also had other cases in which this systolic wave has been less marked, and can quite understand the persistency with which the supporters of the Barclay theory have adhered to their views. I have not the slightest doubt that I have felt the heart contract, in a case of mitral stenosis, during the greater part of the pause and prior to the occur-

rence of the first sound; or, to correct my statement, that I have felt the *right* ventricle contract. I have, however, no evidence to prove that the left ventricle was also in systole. On the contrary, the fact that the wave-like contractile movement commenced shortly after the reduplicated second sound negatives the idea. Dr. Barclay, presupposing that the two sides of the heart were acting in synchronism, concluded that the so-called presystolic murmur was systolic in rhythm. In order to fully appreciate the auscultatory signs resulting from this reduplication of the first sound, we must clearly bear in mind the causes producing that sound. Sherrington informs us (Allbutt, vol. v, p. 467) that it is "found to be due to the vibrations of (*a*) the muscular wall of the ventricles, (*b*) the auriculo-ventricular valves, and (*c*) the mass of blood in the ventricles." Therefore the first sound may be reduplicated in two ways.

1. The two ventricles may not contract synchronously, and that portion of the first sound due to muscle vibrations may be heard during one of the pauses. It partakes of the nature of a burr, and varies in length and intensity in proportion to the amount of asynchronism and the vigor of the heart's action.

2. When the tricuspid and mitral valves are not in tension together the sound resulting from that tension becomes split up into its two component parts, and the text-book reduplication of the first sound is heard. In mitral stenosis the distended condition of the right heart nullifies the sudden tension of the tricuspid valves and prevents its being heard; there is no doubt, however, that any tension in the valves must be antedated and in many cases be synchronous with the previous second sound. The possibility of the reduplication of the second sound being in some cases due to this naturally crosses one's mind, but I do not think it can be entertained. Both these forms of reduplicated first sound may occur in the same patient, the muscle sound and the two valvular sounds being heard alternately for a few beats each. This is by no means common, but I have two cases under treatment in which it is present.

3. In discussing reduplication of the muscle vibratory portion of the first sound, we must bear in miud the nature of the cardiac systole. It is generally accepted that the wave of muscular contraction begins at the base, spreads to the apex, and then returns to the base (Waller's Physiology, pp. 395 and 396), and I believe that "the heart contraction during the pause" described by Barclay is the base-to-apex portion of the right ventricular systole, the first sound occur-

ring when that wave arrives at the apex; the base-to-apex portion being synchronous with the shortened left ventricular systole. On auscultation we hear these muscle vibrations during the diastole of the left ventricle, and they are the cause of the peculiar crescendo character of the so-called presystolic murmur and the intimate blending of the auriculo-systolic portion of it with the first sound. In the later stages of the disease, when there is some failure of compensation, the so-called presystolic murmur is said to be lost; in the majority of these cases, however, the audible muscle vibrations can still be heard, the lost portion being that contributed by the auriculo-ventricular blood current. In many cases this can be restored by rest in bed for a few days, and the characteristic murmur is then again heard. When there is a greater breakdown of compensation we only hear a slapping first sound extending backward into the diastole, the auriculo-systolic and the greater portion of the muscle vibrations being lost.

4. These muscle vibrations also prevent the distinction between the two murmurs of double mitral disease that are heard in the double aortic murmur.

Some authorities are content to deny the occurrence of cases in which it is impossible to tell where the presystolic murmur ends and the systolic begins, and refer to a "soundless" interval, etc.; but these cases do occur, have been shown by me, and require explanation. I maintain that the perfect blending of two murmurs caused by currents traveling in different directions can only result from the introduction of a third element, such as the reduplication of the muscle vibratory portion of the first sound.

5. Whatever view is held regarding the cause of the presystolic murmur, all I think will be agreed that the murmur and the thrill have a common origin. On combining palpation and auscultation this becomes self-evident, the thrill in many cases being felt continuously throughout almost the whole of the diastole and terminating abruptly in the first sound; on the other hand, the thrill in aortic stenosis has a very different character, and makes it difficult to understand how they can both have a common origin, viz., the blood current.

6. Apart from mitral stenosis this form of reduplicated first sound is heard under many other conditions. In children the synchronous action of the two sides of the heart is easily disturbed and a pseudopresystolic murmur heard; this is especially the case if the pericardium is adherent and the contraction of the right ventricle much hampered. We also occasionally have it in mitral regurgita-

tion. The patient when first seen has failure of compensation, the heart's action is much quickened, and pseudopresystolic murmur present; as the heart gains in strength and quiets down, the presystolic element passes off and a pure mitral systolic murmur is heard. This is my own experience, and I presume that of many others.

On the other hand the case may not respond to treatment, but terminate fatally; and if, as occurred recently in the practice of one of our best known men, the diagnosis of mitral stenosis has been made, we are surprised to find at the post-mortem neither contraction nor roughening of the mitral orifice. It is also very common in anemia and imparts a presystolic character to the heart sounds. Other instances might be mentioned, but the above will, I think, show that want of synchronism is by no means so rare as is generally supposed.

In conclusion, I would urge that proper recognition of this result of asynchronism is of the greatest importance in all forms of valvular disease, but essential for the correct diagnosis of mitral stenosis.